

## 9.10.1. Complications of Uveitis (I): Glaucoma

### Introduction

- IOP >24 mm Hg increases the risk of glaucoma
- IOP >30 mm Hg most practitioners tend to treat even without evidence of glaucomatous optic nerve damage
- early IOP elevation with active inflammation is almost always caused by inflammation
  - requires aggressive anti-inflammatory treatment
  - ★ corticosteroid-induced ocular hypertension rarely occurs before 3 weeks
  - ♥ clinicians should resist the urge to prematurely taper corticosteroids because of a fear of corticosteroid-induced ocular hypertension
- unilateral uveitis of sudden onset with open angles and increased IOP may be of infectious origin
  - forward rotation of the ciliary body and lens-iris diaphragm

### Acute secondary angle-closure glaucoma

- choroidal inflammation
  - a presenting sign of VKH syndrome or sympathetic ophthalmia
- pain, elevated IOP, and no posterior synechiae
- UBM or ultrasound evaluation
  - choroidal thickening and anterior rotation of the ciliary body
- treatment
  - aggressive corticosteroid therapy
  - aqueous suppressants
  - cycloplegia
  - peripheral iridotomy or iridectomy is not useful!

### Subacute secondary angle-closure glaucoma

- acute recurrent or chronic anterior segment inflammation
  - circumferential posterior synechiae
  - pupillary block (exclusion of the pupil)
- treatment
  - multiple
    - large
    - exacerbation of inflammation after laser iridotomy
    - intensive topical corticosteroid and cycloplegic therapy
    - iridotomies prone to close
  - peripheral laser iridotomy
    - in patients with brown irides, pretreat the iris with argon laser before using the Nd:YAG laser
    - may lessen the chance of bleeding and facilitate a wider opening
  - surgical iridectomy
    - if laser iridotomy is not successful
    - ± supplemented with goniosynechiolysis

### Chronic secondary angle-closure glaucoma

- chronic intraocular inflammation
  - peripheral anterior synechiae
- treatment
  - topical aqueous suppressants may be inadequate
  - goniosynechiolysis
  - trabeculectomy with mitomycin C
  - glaucoma drainage device

### Acute secondary open-angle glaucoma

- pathogenesis
  - trabecular meshwork is inflamed (trabeculitis)
  - inflammatory debris clogs the angle
- infectious causes of uveitis
  - toxoplasma retinochoroiditis
  - necrotizing herpetic retinitis
  - herpes simplex and varicella-zoster anterior uveitis
  - cytomegalovirus anterior uveitis (including the Posner-Schlossman type)
  - Fuchs heterochromic uveitis (rubella-associated)
- sarcoid uveitis
  - specific treatment of the infectious agent
  - topical cycloplegics and corticosteroids

### Chronic secondary open-angle glaucoma

- direct damage to the trabecular meshwork
- management
  - similar to that of primary open-angle glaucoma
  - strict control of intraocular inflammation through the use of IMT

### Combined-mechanism uveitic glaucoma

- multiple mechanisms may be responsible for elevated pressure in uveitic eyes

### Corticosteroid-Induced Ocular Hypertension and Glaucoma

- corticosteroids in any formulation—topical, peritocular, intraocular, or oral—may induce an elevation of IOP
- fluciclonide intraocular implant need for glaucoma surgery in ~40%
- topical dexamethasone associated with severe and sometimes rapid increases in IOP
- prevention
  - use of a less-potent topical corticosteroid preparation
    - fluorometholone
    - loteprednol
    - rimexolone
  - less frequent administration schedule
    - less likely to induce an IOP elevation
    - less effective in controlling intraocular inflammation

### Management

- first-line agents ★ aqueous suppressants
  - especially when used with IMT and corticosteroids
  - do not exacerbate intraocular inflammation
  - prostaglandin analogues
    - use with caution in eyes with herpetic uveitis
  - the smaller fixed pupil may be at risk for worsening of posterior synechiae
  - causes breakdown of the blood-aqueous barrier
    - pilocarpine should be avoided in uveitis
- greater risk of failure
  - results may be improved by using mitomycin C with intensive topical corticosteroids
  - intense and recurrent inflammation can lead to failure of filtering surgery in uveitic eyes
  - ~80% of patients 1 year after surgery and 62% 5 years after surgery achieve IOP control with 1 or 0 medications
- standard trabeculectomy
  - cataract formation
  - bleb leakage (early and late) could lead to endophthalmitis
  - choroidal effusions
  - excised trabecular block and iris should be submitted for pathologic evaluation
- effective in controlling IOP in ~80% of uveitic eyes x 1 year
  - nonpenetrating deep sclerectomy ± drainage implant
- pediatric uveitis patients
  - goniotomy
    - ~75% chance of reducing IOP to 21 mm Hg or less after 2 surgeries
    - may be complicated by transient hyphema and worsening of the preexisting cataract
  - trabeculectomy
  - trabeculectomy
  - laser sclerostomy
  - visceral sclerostomy
- high rates of failure because of recurrent postoperative inflammation
- higher success rates in a limited number of studies
- most cases of uveitic glaucoma, especially in pseudophakic or aphakic eyes
- anterior chamber
  - tunneled into the vitreous cavity (pars plana)
  - undirectional valve design (valve implant)
  - can prevent postoperative hypotony
- ~75% reduction of IOP from preoperative levels
- 75% of patients achieve target IOP levels with use of 0 or 1 topical antiglaucoma medication after 4 years
- 10%-patient-year
- shallow anterior chamber
- hypotony
- suprachoroidal hemorrhage
- blockage of the drainage device by blood, fibrin, or iris
- CME
- device erosion through the conjunctiva
- valve migration
- drainage device–cornea touch
- corneal decompensation
- retinal detachment
- may worsen ocular inflammation
- hypotony
- phthisis
- generally considered ineffective in eyes with uveitis
- laser trabeculoplasty